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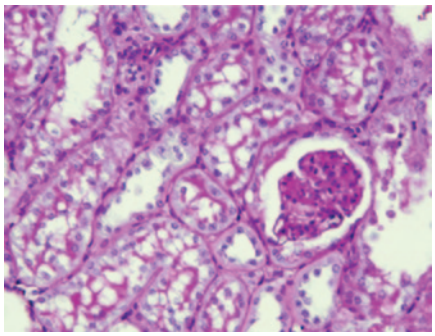
Chronic kidney disease worsens sepsis



The presence of chronic kidney disease (CKD) has an adverse effect on other illnesses. In their recent study, Leelahavanichkul *et al.* generated a mouse model of sepsis by cecal ligation. As expected, sepsis resulted in kidney and liver damage. However, the damage was more severe when sepsis was induced in mice with CKD. The kidney eliminates several cytokines (by excretion or metabolism), which could be the mechanism of aggravation of sepsis. Thus, the authors measured the accumulation of several proinflammatory cytokines, including high-mobility group box protein-1 (HMGB1), vascular endothelial growth factor (VEGF), tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and IL-10, in various conditions and found that, while their levels were increased in CKD, the levels in sepsis plus CKD were higher than can be accounted for by decreased renal clearance. Antisera against HMGB1 attenuated the effect of sepsis but did not prevent it. **See page 1198.**

Worse prognosis for those who develop acute kidney injury in the setting of chronic kidney disease

As they report in this issue, Wu *et al.* examined the records of more than 9000 patients in Taiwan who had undergone major surgery. They found that those who had CKD with an estimated glomerular filtration rate less than 45 ml/min and then developed acute kidney injury (AKI) had worse survival after discharge and follow-up for nearly 5 years. Their hazard ratio was three times that of patients who developed AKI but had no CKD before surgery. Further, those who had AKI in the setting of CKD had a dialysis incidence of 22 per 100 person-years compared with 0.17 in the patients who had AKI with normal renal function. Similarly, those with CKD had a higher hazard ratio for mortality. These studies emphasize the worse prognosis of patients with CKD who develop AKI after major surgery. **See page 1222.**

Inflammatory markers predict incident chronic kidney disease

Increasing evidence suggests that an inflammatory state is at the core of many illnesses previously not thought to have an inflammatory component. Shankar *et al.* studied the association of inflammatory markers such as C-reactive protein levels, white blood cell counts, and TNF-α receptor and IL-6 levels in a population cohort of almost 5000 people. They found that all of these parameters were associated with the prevalence of CKD in cross-sectional analysis. When they performed longitudinal studies examining those with normal renal function at the outset, TNF-α receptor, white blood cells, and IL-6, but not C-reactive protein, were positively correlated with incident CKD. **See page 1231.**